Brit. Heart J., 1968, **30**, 591.

The 'A' Wave of the Apex Cardiogram in Aortic Valve Disease and Cardiomyopathy

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Marey (1863) introduced a graphic method of recording the apex beat and showed that the main systolic impulse was immediately preceded by a small wave due to atrial systole. Potain (1875) found that the atrial systolic wave was greatly increased in hypertensive heart disease, that it could be palpated, and was usually associated with a presystolic (fourth) heart sound. Barlow and Kincaid-Smith (1960), Grayzel (1960), and Parry and Mounsey (1961) have recently drawn attention to the presence of a palpable atrial impulse at the apex in hypertensive heart disease. Large palpable atrial waves giving rise to a double apical impulse have also been described in hypertrophic obstructive cardiomyopathy and they have been demonstrated graphically by means of the apex cardiogram (Boiteau and Allenstein, 1961; Benchimol, Legler, and Dimond, 1963; Cohen *et al.*, 1964; Braunwald *et al.*, 1964; Wigle, 1964; Tafur, Cohen, and Levine, 1964; Wolfe, 1966; Nagle *et al.*, 1966).

We have also been impressed by the readily palpable atrial impulse in both obstructive and non-obstructive cardiomyopathy. In our experience a palpable atrial impulse is also a relatively common finding in other types of left ventricular disease, particularly in aortic stenosis and aortic regurgitation. Since it is an easily detectable physical sign we felt it worth while to assess its significance.

The purpose of this paper is to examine the relation between left heart pressure, the "A" wave of the apex cardiogram, and palpation of the apex beat in a group of patients with left ventricular disease. This study is confined to patients with aortic valve disease and cardiomyopathy.

Patients and Methods

A total of 66 patients forms the main basis of this study and they are subdivided as follows.

Received December 5, 1967.

(1) *Aortic Stenosis*—29 patients (25 valvar; 3 subvalvar; 1 supravalvar).

(2) *Aortic Stenosis and Regurgitation*—10 patients.

(3) *Aortic Regurgitation*—12 patients.

(4) *Cardiomyopathy*—15 patients (10 obstructive; 5 idiopathic).

All the patients were in normal sinus rhythm, and mitral valve disease was excluded on the basis of clinical, haemodynamic, and operation findings. A full clinical examination and routine radiological and electrocardiographic studies were carried out on each patient. Special attention was paid to palpation of the cardiac impulse. The character of the main systolic impulse was noted, and a presystolic atrial beat was specifically sought for, with the patient lying on the left side. Third and fourth heart sounds were carefully listened for during auscultation.

A complete phonocardiographic study was done on each patient, and apex cardiograms were recorded by methods previously described (Coulshed and Epstein, 1963). Special care was taken to ensure that the piezoelectric crystal microphone used for recording the apex cardiogram had an adequate time constant ($>1{\cdot}0$ second), as described by Roberts and Jones (1963).

The height of the "A" wave of the apex cardiogram was measured from its onset to its peak and expressed as a percentage of the total amplitude of the tracing (Fig. 1), as described by Benchimol and Dimond (1962). Between 3 and 6 complexes were measured and an average figure was derived.

Combined right and left heart catheterization was performed for diagnostic purposes in all patients. Left atrial and left ventricular catheterization was performed by transseptal puncture of the atrial septum. A second catheter was inserted percutaneously into the femoral artery and passed to the aortic root and, where possible, into the left ventricle.

Pressures were recorded with the breath held in partial expiration using equisensitive induction manometers and a multichannel oscilloscopic recorder (Cambridge Instrument Company). The zero baselines were set at 5 cm. below the sternal angle. In 64 patients left ventricular and aortic pressures were recorded simul-

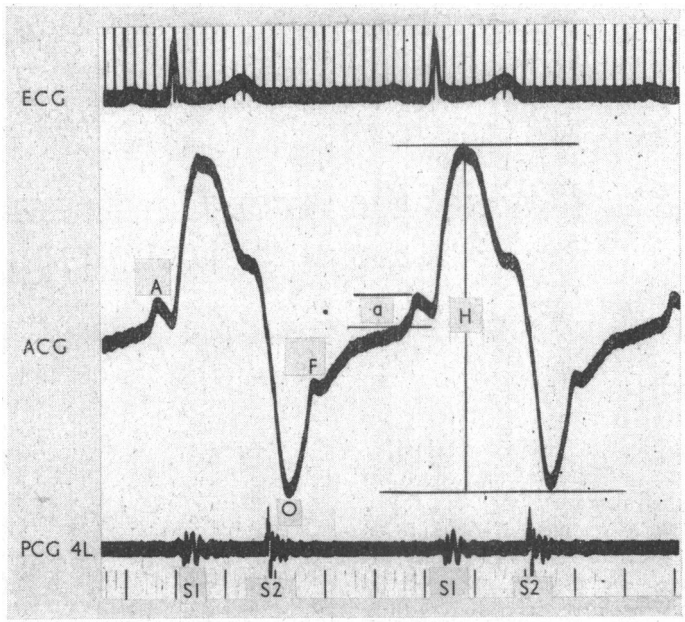

FIG. 1.—The normal apex cardiogram (ACG) with simultaneous phonocardiogram (PCG) and electrocardiogram (ECG). "A" is the atrial systolic wave: "O" occurs at the time of opening of the mitral valve: "F" is the end of rapid early filling: "H" is the total height of the apex cardiogram from point "O" to peak. "a" is the height of the "A" wave measured from its onset to peak. The height "a" expressed as a percentage of "H" is known as the a/H ratio. S1=the first heart sound; S2=the aortic second sound.

taneously, and in one patient a pullback tracing across the aortic valve was obtained. In one patient left ventricular pressure was not recorded. Left ventricular diastolic pressures were always recorded at high sensitivity for accuracy of measurement. In 16 patients simultaneous left atrial and left ventricular pressures were recorded, and in 47 patients adequate pullback tracings across the mitral valve were obtained so that mitral valve disease could be excluded. In 3 patients no direct left atrial pressure was obtained, but in 2 of these patients pulmonary capillary wedge pressures were recorded. The diastolic phases of the left ventricular and left atrial tracings matched each other in all the patients studied. Pressure measurements were made from at least 3 separate cycles and an average figure was derived. Measurements were made of left ventricular end-diastolic pressure; mean left atrial pressure; the pressure at the peak of the left atrial "a" wave; and of the height and duration of the left atrial "a" wave from its onset to its peak.

A group of 18 patients had left atrial or left ventricular pressures recorded simultaneously with the apex cardiogram. Of these patients, 14 were from the 66 forming the basis of this study. Records were also available from 4 other patients not included in the study: 2 patients with coarctation of the aorta, 1 with hypertension, and 1 with aortic regurgitation. These patients were studied to assess the direct relation between haemodynamics and the apex cardiogram.

Cine-angiography was performed in 48 patients by left atrial, left ventricular, or aortic injection of contrast medium. Some patients had more than one cine-angiographic study. In 4, left-sided cine-angiograms, taken in the right anterior oblique projection at a speed of 50 frames per second, were analysed serially. The contrast medium was injected into the left atrium in 2 of the patients and into the left ventricle in 1 patient. In the other patient, aortic regurgitation produced good opacification of the left ventricle following aortic root injection of radio-opaque dye. The image was projected onto the screen of an analytical projector (Tage-Arno; Copenhagen), and an orthodiagram of the dye-filled left ventricle was drawn from each cine frame. At least 3 cycles were studied and care was taken to exclude cycles disturbed by premature beats. Both the area and the volume of each frame were calculated in arbitrary units and then plotted onto a graph in time sequence. The cyclic changes were indirectly related to the simultaneous electrocardiogram and also to a closely related left atrial or left ventricular pressure pulse.

The 4 patients so studied were No. 5 and No. 29 with aortic stenosis, No. 41 with aortic regurgitation, and No. 61 with obstructive cardiomyopathy. In the 2 patients with aortic stenosis (No. 5 and 29), who had left atrial injection of dye, area and volume calculations were made on the left atrium as well as on the left ventricle.

RESULTS

Normal Controls (30 subjects). A normal apex cardiogram is illustrated in Fig. 1. The percentage

amplitude of the "A" wave (a/H ratio) in this group of 30 subjects averaged 7·0 per cent, with a standard deviation of ±2·25 per cent (range 3·8 to 12·0%). The duration of the "A" wave did not exceed 60 milliseconds.

The essential clinical and haemodynamic data on the 66 patients are set out in Tables I, II, and III.

Relation of a/H Ratio to Left Heart Pressures.

Left Ventricular End-diastolic Pressure and a/H Ratio. This is shown graphically in Fig. 2 for all 66 patients. There was a tendency for the a/H ratio to increase with the left ventricular end-diastolic pressure. With 3 exceptions (all with aortic stenosis) an a/H ratio above 16 per cent was associated with a pressure of 11 mm. Hg or more. Only 3 patients had a left ventricular end-diastolic pressure above 11 mm. Hg, with a normal a/H ratio.

Left Atrial Peak "a" Wave Pressure and a/H Ratio. This is shown graphically in Fig. 3. The a/H ratio in general increased as the peak "a" wave pressure rose. With 3 exceptions (the same 3 patients with aortic stenosis as above) an a/H ratio above 16 per cent was associated with a peak "a" wave pressure of 12 mm. Hg or above. Only 3 patients had a peak "a" wave pressure above 12 mm. Hg, with a normal a/H ratio.

Left Atrial "a" Wave Pulse Pressure and a/H Ratio. This is shown graphically in Fig. 4. The a/H ratio in general increased as the "a" wave pulse pressure increased. With 4 exceptions (3 with aortic stenosis, 1 with obstructive cardiomyopathy) an a/H above 16 per cent was associated with an "a" wave pulse pressure of 4·5 mm. Hg or above. There were, however, 8 patients with a normal a/H ratio and an "a" wave pulse pressure above 4·5 mm. Hg.

Simultaneous Apex Cardiogram and Left Heart Pressures. The relation between the a/H ratio and simultaneous left heart pressure is shown graphically in Fig. 5 and illustrated in Fig. 6. There was a significant positive correlation between the a/H ratio and peak "a" wave pressure (correlation coefficient $r = 0{\cdot}60$; $p < 0{\cdot}01$). An a/H ratio of 14 per cent or more was always associated with a peak "a" wave pressure of at least 12 mm. Hg. When the a/H ratio was 20 per cent or more, the peak "a" wave pressure exceeded 20 mm. Hg.

The left atrial "a" wave pulse pressure and the a/H ratio also showed a significant positive correlation of lesser degree ($r = 0{\cdot}45$; $p < 0{\cdot}05$). An a/H ratio of more than 18 per cent was associated with an "a" wave pulse pressure of 5 mm. Hg or more.

TABLE I

AORTIC STENOSIS

Patient No., age, and sex	Palpable atrial impulse	a/H ratio (%)	Left atrial pressure (mm. Hg)			Left ventricle (mm. Hg)		LV/aorta systolic gradient (mm. Hg)	Fourth heart sound	
			"a" wave peak	"a" pulse pressure	Mean	"a" wave peak	End-diastolic pressure		Auscultation	Phonocardiogram
1 14 M	−	12·5	11·5	3·5	—	11	8	75	−	−
2 17 F	−	7	9	6·5	6·5	10	9	110	−	+
3 16 M	−	8	9	3	7	10	9	100	−	−
4 17 M	−	6	8	3	7·6	8	7	55	−	−
5 69 F	+	28	23	11·5	12·5	20	20	75	−	+
6 45 M	+	19	15·5	2	12	14	12	90	−	+
7 35 M	+	39	9	3	7	9	7·5	130	+	+
8 52 M	−	20	7·5	2	6·5	10	9	85	+	+
9 63 M	+	15·5	6·5	3·5	4·5	10·5	8	95	+	+
10 34 F	−	9·5	6·5	1·5	6	8	7·5	50	−	−
11 54 F	+	13	8	6	5	8	5	80	+	+
12 54 M	−	10	5	2·5	3	6	5	60	−	−
13 25 M	+	16	11·5	4	8	11	9	130	−	−
14 24 M	−	11	8	5	3·5	9	8	100	−	−
15 26 M	−	11	10	4	8	11	10	60	−	−
16 59 M	−	7	10	5	5	10	8	95	−	+
17 45 M	+	27	13·5	4·5	9	14·5	14	110	+	+
18 46 M	+	19	24	6	21	24	21	105	−	−
19 17 F	−	7·5	7·5	2	6·5	8	7	25	−	−
20 51 M	+	34	10	5	6	10	9	95	−	+
21 60 F	+	22	13	7·5	6	12	10	115	+	−
22 33 M	+	16·5	15	7	12	17	15	100	+	+
23 49 F	+	17	13·5	4·5	11	13	11	105	+	−
24 46 M	−	8	14·5	5·5	9	14	10	110	−	+
25 58 M	+	28	13·5	7·5	9	16·5	16	75	+	+
26 55 M	+	16	18	6·5	14	22	20	60	+	+
27 44 F	+	27	15	7·5	8	14	12	80	+	−
28 59 M	−	9·5	12	5·5	7·5	12	9	95	−	−
29 23 M	−	9	13	3	11·5	14	11·5	90	−	−

TABLE II

AORTIC STENOSIS AND REGURGITATION, AND AORTIC REGURGITATION ALONE

Patient No., age, and sex	Palpable atrial impulse	a/H ratio (%)	Left atrial pressure (mm. Hg)			Left ventricle (mm. Hg)		LV/aorta systolic gradient (mm. Hg)	Fourth heart sound	
			"a" wave peak	"a" pulse pressure	Mean	"a" wave peak	End-diastolic pressure		Auscultation	Phonocardiogram
Aortic Stenosis and Regurgitation										
30 48 M	+	11·5	10	5·5	5	13	11·5	60	+	+
31 41 M	+	21	18	12	13·5	22	20	25	+	+
32 48 M	+	23	14	10	5	14	12	50	+	+
33 38 F	+	13·5	15	7	11	15	14	55	−	−
34 56 F	+	24	32	15	14	28	20	105	+	−
35 37 M	+	36	22	8·5	19	20	15	85	+	+
36 54 F	+	20	14	8	8	14	12	35	+	+
37 63 M	+	18	22	12	20	22	19	30	−	+
38 44 M	+	25	25	13	19	24	15	110	+	+
39 40 M	+	16	15·5	10	11	15	14	90	+	+
Aortic Regurgitation										
40 27 F	+	10	13	6	5	12	8	0	+	−
41 21 M	+	17·5	15	6·5	8	15	15	0	+	−
42 42 M	+	30	21	10	13	23	21	0	+	−
43 52 F	+	13	26	8·5	23	30	26	20	−	−
44 31 M	+	13	13	3	9	14	13	0	+	+
45 27 M	+	20	12	5	8	12	12	0	+	+
46 50 M	+	14	8	4	4	8	7·5	0	+	+
47 48 M	+	37	20	7	19	—	—	—	+	+
48 54 M	+	16	10	2·5	7	14	13	40	+	+
49 36 M	+	36	23	10	20	24	23	0	−	+
50 43 F	+	16	7	3·5	4	8	6	5	+	+
51 26 M	+	15	12	5	8	12·5	12	0	+	+

TABLE III

CARDIOMYOPATHY

Patient No., age, and sex	Palpable atrial impulse	a/H ratio (%)	Left atrial pressure (mm. Hg)			Left ventricle (mm. Hg)		LV/aorta systolic gradient (mm. Hg)	Fourth heart sound	
			"a" wave peak	"a" pulse pressure	Mean	"a" wave peak	End-diastolic pressure		Auscultation	Phonocardiogram
Obstructive Cardiomyopathy										
52 45 M	+	33	25	13	12	25	25	60	+	+
53 37 F	+	32	—	13 (LV)	—	32	30	60	+	+
54 21 F	+	23	12·5	4	7	13	12·5	100	−	−
55 41 M	+	24	30	23	20	30	28	100	−	+
56 20 F	−	10·5	12	4·5	11	12	8	35	−	−
57 48 F	+	29	31	10	27	25	24	110	+	+
58 27 M	+	30	16	9·5	9	18	17	20	+	+
59 25 M	−	13	9	2	8	13	12	95	+	+
60 28 M	+	17	14	7	9	15	14	15	+	+
61 38 M	+	26	38	19	24	43	39	145	+	+
Idiopathic Cardiomyopathy										
62 22 F	−	34	19	8·5	12	20	18	0	+	+
63 55 F	+	32	16	8	10	16	15	0	+	+
64 35 F	+	18	21	12	27	18	17	0	+	+
65 53 F	+	7·5	10	6	4	10	8	0	+	+
66 46 M	+	27	16	7	13	16	14	0	+	+

Relation of a/H Ratio to Findings on Palpation. Fig. 7 shows the relation between the findings on palpation and the a/H ratio on the apex cardiogram. When the a/H ratio was 14 per cent or more the "a" wave was palpable, with only 2 exceptions. On the other hand, 3 patients had a palpable "a" wave with an a/H ratio below 12 per cent.

Discussion

Attention has recently been drawn to graphic methods of recording the atrial impulse at the cardiac apex by means of the kinetocardiogram (Harrison, Coghlan, and Prieto, 1961), the apex cardiogram (Benchimol and Dimond, 1962), or the impulse cardiogram (Beilin and Mounsey, 1962).

The "A" wave of the apex cardiogram is due to displacement of the wall of the left ventricle in late diastole by left atrial systole (Mackenzie, 1902). It appears early if the P–R interval is prolonged, it is dissociated from the ventricular complexes in complete heart block, and it disappears with the onset of atrial fibrillation (Coulshed and Epstein, 1963). The "A" wave of the apex cardiogram coincides

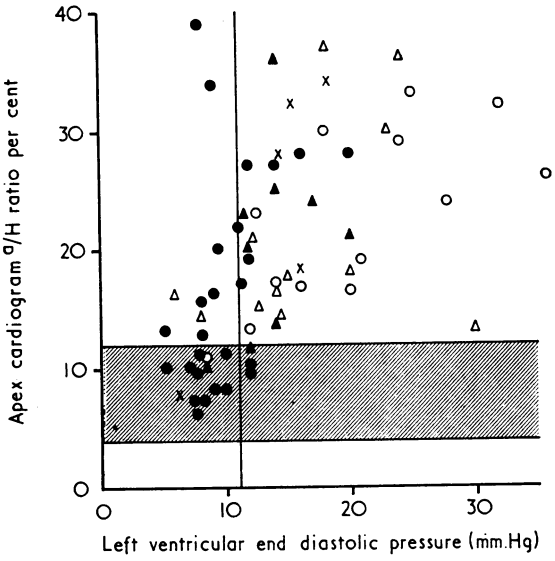

FIG. 2.—The a/H ratio compared with left ventricular end-diastolic pressure. The shaded area in this and subsequent figures represents the normal range (7·0% ± 2 SD). The following symbols apply to all figures. ●—Aortic stenosis; ▲—aortic stenosis and regurgitation; △—aortic regurgitation; ○—hypertrophic obstructive cardiomyopathy; ×—idiopathic cardiomyopathy.

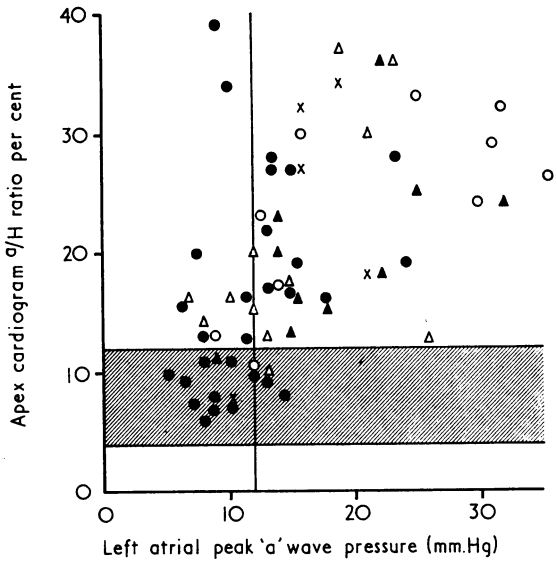

FIG. 3.—The a/H ratio compared with left atrial peak "a" wave pressure.

with the fourth heart sound (see Fig. 9 and 12), and follows the P wave of the electrocardiogram at a definite time interval (Hartman and Snellen, 1960).

Simultaneous records of left atrial or left ventricular pressure with the apex cardiogram show that the intracardiac "a" wave pressure pulse coincides with the "A" wave of the apex cardiogram (Fig. 6).

Benchimol and Dimond (1962) first drew attention to the increase in left ventricular end-diastolic pressure or pulmonary capillary wedge pressure in association with tall "A" waves on the apex cardiogram in patients with arteriosclerotic heart disease, and similar observations were made by Rörvik (1963). Exercise increased both left ventricular end-diastolic pressure and the amplitude of the "A" wave of the apex cardiogram (Dimond and Benchimol, 1963).

Tall "A" waves have been demonstrated by apex cardiography in patients with obstructive cardiomyopathy (Benchimol *et al.*, 1963; Wigle, 1964; Braunwald *et al.*, 1964; Tafur *et al.*, 1964; Wolfe, 1966; Nagle *et al.*, 1966) and also in aortic stenosis (Goldblatt, Aygen, and Braunwald, 1962: Tafur *et al.*, 1964; Tavel *et al.*, 1965; Wolfe, 1966).

Increased left atrial activity is a well-recognized finding in left ventricular disorders and it can be demonstrated in several ways. A fourth heart sound is frequent and enlargement of the left atrium may be demonstrated radiologically, and also electrocardiographically by broadening of the "P" waves. Necropsy studies in left ventricular disease show that left atrial volume is increased and the muscle fibres

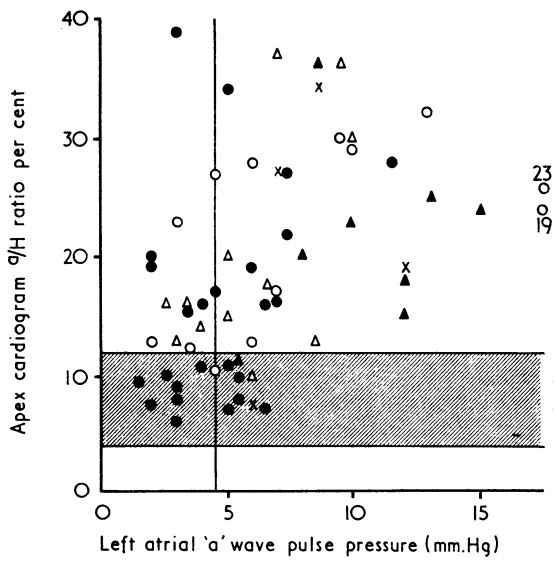

FIG. 4.—The a/H ratio compared with left atrial "a" wave pulse pressure.

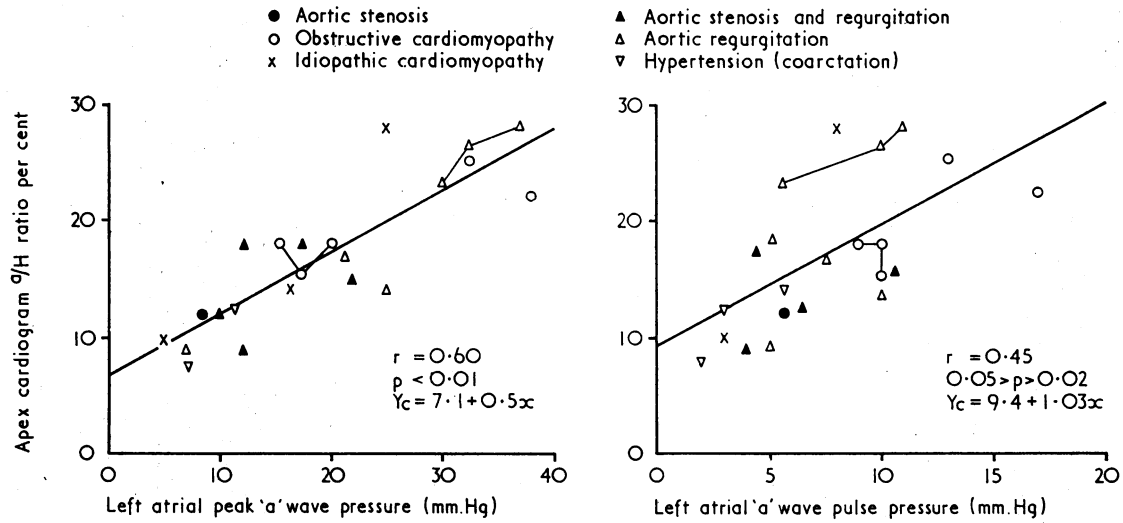

FIG. 5.—Left atrial pressure recorded simultaneously with the apex cardiogram. Twenty-two observations in 18 patients. Lines join different observations on the same patient. Left atrial peak "a" wave pressure and left atrial "a" wave pulse pressure have each been plotted against the a/H ratio. The correlation coefficient r, the p factor, and the regression equation are shown on each graph together with the calculated regression line.

hypertrophied (Cobbs, Shillingford, and Steiner, 1957). Symptoms of left heart failure in left ventricular disorders imply that left atrial pressure is increased, and this has been confirmed by cardiac catheter studies (Selzer and McCaughey, 1960). Tall left atrial "a" waves due to increased vigour of left atrial contraction have been shown by direct measurement of left-sided heart pressures in various types of left ventricular disease (Braunwald and Frahm, 1961).

Ventricular distensibility is altered in left ventricular disease due to hypertrophy of the left ventricular wall. The left ventricular end-diastolic pressure rises in association with a rise in left atrial pressure. Braunwald and Frahm (1961) have shown that the left atrium is of considerable haemodynamic importance in patients with raised left ventricular end-diastolic pressure and sinus rhythm. The left atrial contraction is more vigorous and prolonged, enabling the end-diastolic pressure in the left ventricle to be raised considerably above mean left atrial pressure. It thus allows the left ventricle to contract from a larger end-diastolic fibre length, and contributes to ventricular performance with minimal rise in mean left atrial pressure.

The transfer of a given volume of blood to the left ventricle in diastole due to left atrial systole will produce relatively greater outward ventricular wall motion in a small chamber than in a large chamber.

The left atrial pressure required to produce a given volume change in the ventricle will depend on the distensibility (compliance) of the ventricle. With diminished ventricular compliance there will be large increments of pressure with small increases of volume. A normal left atrial stroke volume can only be accommodated in a non-compliant ventricle if the filling pressure is raised. Dodge, Hay, and Sandler (1962) have shown the relation between left ventricular diastolic pressure and volume in various cardiac disorders. In aortic stenosis and obstructive cardiomyopathy where the diastolic left ventricular cavity is small and incompliant, the pressure volume curve shows a steep rise even though the diastolic volume in the cavity is not greater than normal. In aortic regurgitation there is a large ventricular end-diastolic volume. The pressure volume relation may be within normal limits over the initial part of ventricular filling when a normal amount of blood is easily accommodated without any rise of filling pressure. Towards the end of diastole when the volume is large the ventricle becomes increasingly non-distensible and a normal atrial stroke volume may then produce a considerable rise in ventricular diastolic pressure. Thus atrial systole may act on both small and large ventricles over the poorly distensible (incompliant) part of their diastolic pressure-volume curve.

It has been suggested that passive filling of the left ventricle is diminished in left ventricular disease due to the resistance offered by the ventricle. The

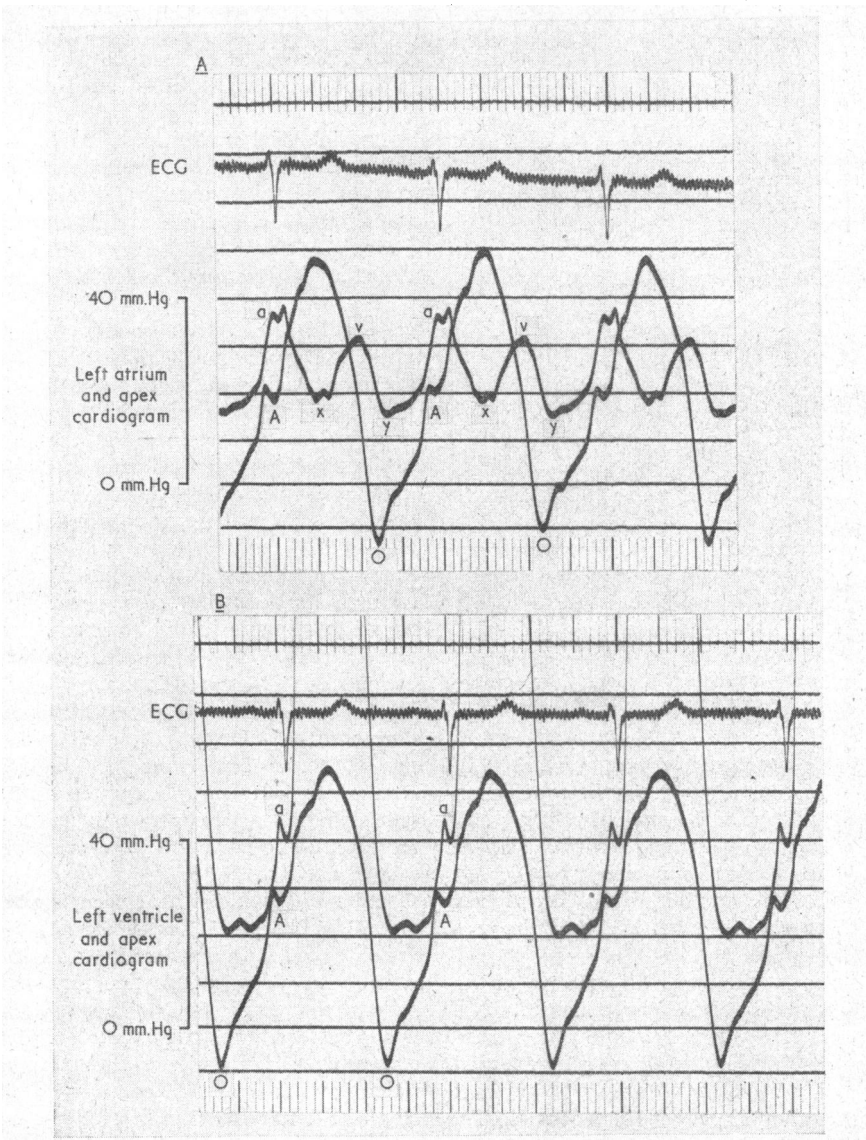

FIG. 6.—Patient No 61. Obstructive cardiomyopathy. Systolic gradient 145 mm. Hg. Palpable atrial impulse and fourth heart sound: a/H ratio 26 per cent. Apex cardiogram recorded simultaneously with (a) left atrial pressure and (b) left ventricular diastolic pressure. The "A" wave of the apex cardiogram precedes the "a" wave of the pressure pulse due to transmission delay in the catheter-manometer system.

contribution of atrial systole to filling may therefore be increased (Braunwald *et al.*, 1963).

The findings in this study show that a large apex cardiogram "A" wave (a/H ratio) is usually associated with a rise in left ventricular end-diastolic pressure (Fig. 2), left atrial peak "a" wave pressure (Fig. 3), and left atrial "a" wave pulse pressure (Fig, 4). Although there is a positive correlation between the a/H ratio of the apex cardiogram and peak left atrial "a" wave pressure when they are recorded simultaneously, it is not a very close relation (Fig. 5). This is not surprising since the apex cardiogram primarily reflects changes in volume rather than changes in pressure. The size of the "A" wave on the apex cardiogram depends on the amount of displacement of the left ventricular wall during atrial systole. A large "A" wave therefore implies an increased atrial stroke volume as well as a more forceful atrial contraction.

Cine-angiographic Studies. From the total of 66 patients in the series, 48 cine-angiograms with ade-

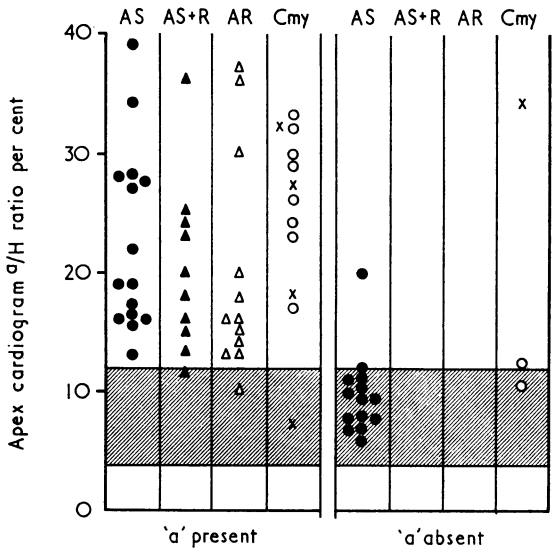

FIG. 7.—The a/H ratio compared with the presence or absence of a palpable atrial impulse for each of the four main groups of patients. AS—aortic stenosis; AS + R—aortic stenosis and regurgitation; AR—aortic regurgitation; Cmy—cardiomyopathy.

quate detail of the left ventricle were available. In 41 there was a prominent atrial systolic contraction wave, following passive left ventricular filling and immediately preceding ventricular systole, which produced a sharp outward movement of the left ventricular wall. This motion of the ventricular wall was easily appreciated when the cine-film was projected at high speed.

In normal subjects the atrial filling wave in the left ventricle was small and inconspicuous.

Four patients had detailed single-plane cine-angiographic studies. Left atrial systole increased the left ventricular diastolic volume by 18 per cent in patient No. 5 and by 22 per cent in patient No. 29, both with aortic stenosis. Atrial systole contributed 12·5 per cent of ventricular filling in patient No. 41, with aortic regurgitation, and 12·0 per cent in patient No. 61, with obstructive cardiomyopathy (Table IV and Fig. 8). The contribution of atrial systole was relatively greater when expressed as a percentage of ventricular stroke volume (Table IV).

Physiological studies in animals have shown that left atrial systole may contribute from 15 to 60 per cent of the total volume of left ventricular filling (Gesell, 1916; Wiggers and Katz, 1922; Jochim, 1938). At rapid heart rates atrial systole is responsible for a larger proportion of left ventricular filling. It becomes maximal when the T-P interval disappears and left atrial systole coincides with the phase of rapid filling (Gribbe *et al.*, 1959). There are few physiological studies in man of the contribution of the left atrium to ventricular filling. Samet, Castillo, and Bernstein (1966) studied 20 normal subjects using sequential atrial and ventricular pacing. A correctly timed atrial systole increased ventricular stroke volume by 16 per cent. Martin and Cobb (1966) and Carleton and Graettinger (1967) found by artificial pacing that atrial systole contributed approximately 20 per cent of left ventricular stroke volume in the absence of valve disease. Kroetz *et al.* (1967) studied atrial function in patients with aortic stenosis by artificial pacing. Instantaneous recovery of effective atrial contraction increased the stroke volume index by an average of 28 per cent (range 14 to 55%). During studies of steady state integrated cardiac performance effective atrial contraction had less effect and increased the cardiac index by an average of 13 per cent (range 0 to 31%).

Atrial function has also been studied by radiological techniques. Gribbe *et al.* (1959) studied cyclic changes in left atrial and left ventricular volume in dogs using cine-angiography. They showed that about 60 per cent of atrial emptying was passive and mainly in early diastole, and the remaining 40 per cent was due to active atrial contraction. Atrial systole contributed about 15 per cent of the left ventricular stroke volume. Grant, Bunnell, and Greene (1964) estimated from biplane angiograms that atrial stroke volume was about 20 per cent of left ventricular stroke volume in normal subjects. In patients with aortic stenosis, left atrial

TABLE IV

CONTRIBUTION OF ATRIAL SYSTOLE ESTIMATED BY CINE-ANGIOGRAPHY

Patient No.	Diagnosis	Contribution of atrial systole to LV filling (%)		Contribution of atrial systole to LV stroke volume (%)	
		Area	Volume	Area	Volume
5	Aortic stenosis	12·5	18	14	19
29	Aortic stenosis	19	22	27	24
41	Aortic regurgitation	6·6	12·5	13	17·5
61	Obstructive cardiomyopathy	10·5	12	16	14

Note: The figures in the table are all percentages and are the mean values for three successive cardiac cycles.

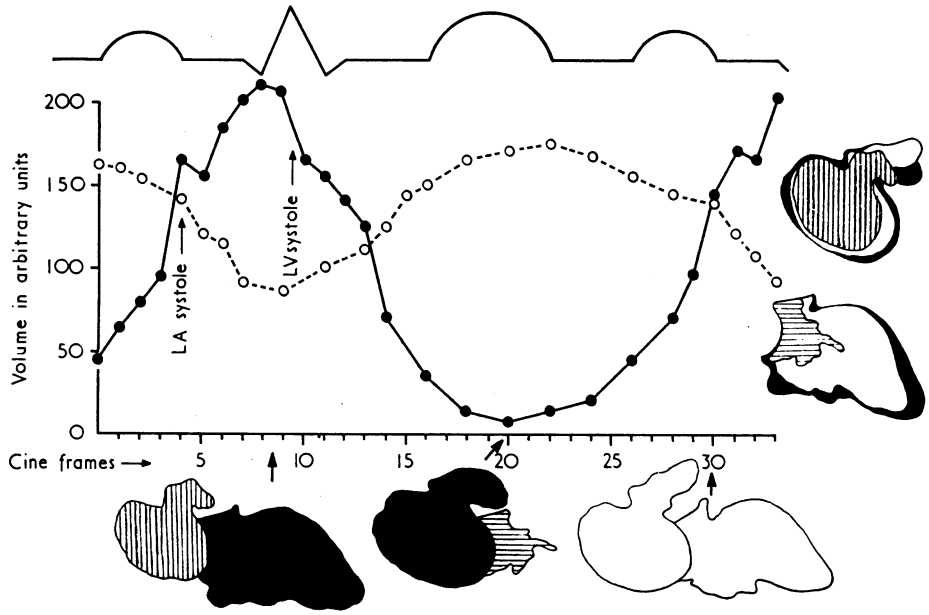

FIG. 8.—Patient No. 5. Aortic stenosis. Systolic gradient 75 mm. Hg; a/H ratio 28 per cent. Relative changes in left atrial volume (dashed line) and left ventricular volume (black line) estimated by the method of Greene *et al.* (1967), from the right anterior oblique cine-angiogram during one complete cycle using frame by frame analysis (50 frames per second). No correction has been made for image magnification. LV volume increases as LA volume diminishes. Following LA systole there is an increase in LV volume of 19 per cent coincident with a rapid fall in LA volume. LV volume is minimal at end-systole when LA volume is maximal.

Along the bottom are three simultaneous LA and LV silhouettes at three phases in the cycle shown by the broad arrows. The first just before ventricular systole; the second at end-systole; the third at the end of passive ventricular filling just before atrial systole. Areas shaded black are at maximal volume; cross-hatched areas are at minimal volume; non-shaded areas are just before atrial systole.

On the right hand side the three phases for each chamber have been superimposed. The black shaded area on the superimposed ventricular silhouettes represents the increase in volume produced by atrial systole.

stroke volume averaged 40 per cent of left ventricular stroke volume, and hence the atrium made a substantially larger contribution to left ventricular filling. However, these workers used the term "left atrial stroke volume" to refer to the total change in left atrial volume throughout the cardiac cycle and not to the volume of blood transferred to the ventricle by atrial contraction. Hawley, Dodge, and Graham (1966) found, on biplane angiograms, that the cyclical left atrial volume change was on average 27 per cent of left ventricular stroke volume in aortic valve disease and 46 per cent in cardiomyopathy. No estimate was made of the proportion of left ventricular filling due to left atrial systole. The findings of these two groups of workers are therefore essentially similar in that they show an increased atrial transport function in left ventricular disease but without distinguishing between the passive and the active components.

Studies of left atrial volume changes in two of our patients with aortic stenosis (No. 5 and 29) showed that about half of atrial emptying was passive and that half was due to atrial systole (Fig. 8). These findings are similar to those reported by Gribbe *et al.* (1959) in dogs. If these observations on passive and active atrial function are applied to the data of Grant *et al.* (1964), then atrial systole contributes about 10 per cent of left ventricular stroke volume in normal subjects and 20 per cent in patients with aortic stenosis. We have not studied any normal subjects but our findings in aortic stenosis are similar to those of Grant *et al.* (1964) (Table IV). There is, therefore, an increased left atrial contribution to ventricular filling in patients with left ventricular disease.

Apex Cardiogram and Palpation of "A" Wave. The a/H ratio in normal subjects measured 7 per cent ±2·25 per cent. These findings are similar to those of Benchimol and Dimond (1962) (7·8% ±1·4%) and of Rios and Massumi (1965) (8% ±2·5%).

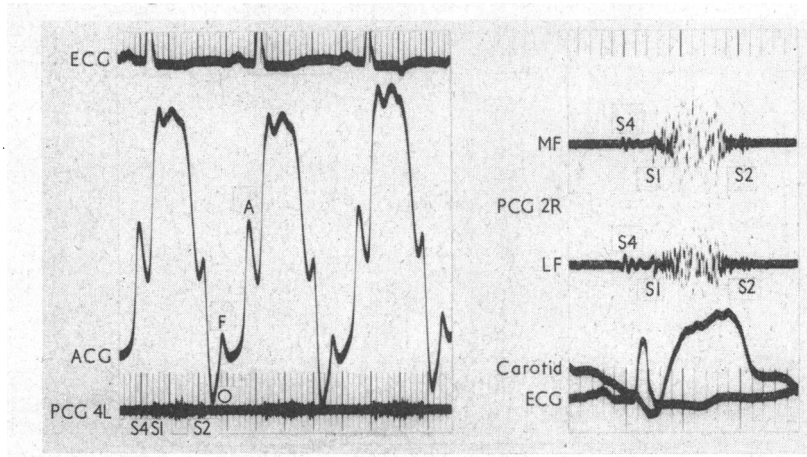

FIG. 9.—Patient No. 7. Severe calcific aortic stenosis. Peak systolic gradient 130 mm. Hg. Palpable atrial impulse. Audible fourth heart sound. The apex cardiogram shows a tall "A" wave; a/H ratio 39 per cent. The phonocardiogram shows an anacrotic carotid pulse with a mid-systolic ejection murmur and a fourth heart sound (S4) coinciding with the "A" wave of the apex cardiogram.

The "A" wave cannot be palpated in normal subjects because of its small amplitude. In left ventricular disease disordered left ventricular function imposes a haemodynamic burden on the left atrium and the "A" wave increases in size and may become palpable (Fig. 9). Whether it becomes palpable or not depends on several factors. The configuration of the chest wall is clearly of great importance. Heavy musculature, obesity, large breasts, and emphysema make it difficult to feel. Palpation in the left lateral position may help by allowing closer contact between the heart and the chest wall.

If the P–R interval is short and left atrial systole is prolonged, ventricular contraction may begin towards the end of atrial systole before atrial relaxation has commenced. The peak of the "A" wave then merges with the upstroke of the apex cardiogram, and it may be difficult to palpate since it is not sufficiently separated from the ventricular systolic impulse (Fig. 10). This occurred in 5 of our patients and the "A" wave was not palpable in 2 of them despite its large size. Occasionally the systolic apical thrust has a bifid form, especially, but not exclusively, in obstructive cardiomyopathy. A double impulse may then be palpable and the first component mistaken for a large "A" wave (Fig. 11). In some patients it is even possible to feel 3 impulses, i.e. the "A" wave and the bifid systolic impulse.

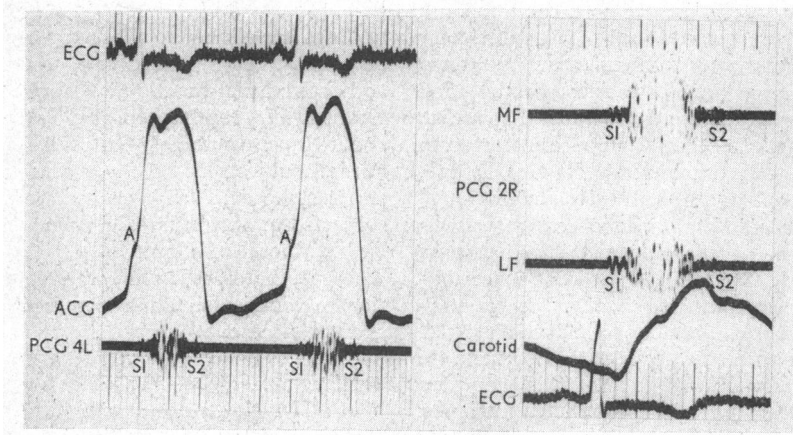

FIG. 10.—Patient No. 8. Severe aortic stenosis. Peak systolic gradient 85 mm. Hg. No clearly palpable atrial impulse. The apex cardiogram shows a tall "A" wave closely applied to the upstroke of the main systolic wave: a/H ratio 20 per cent. The PCG shows an anacrotic carotid pulse and a mid-systolic ejection murmur.

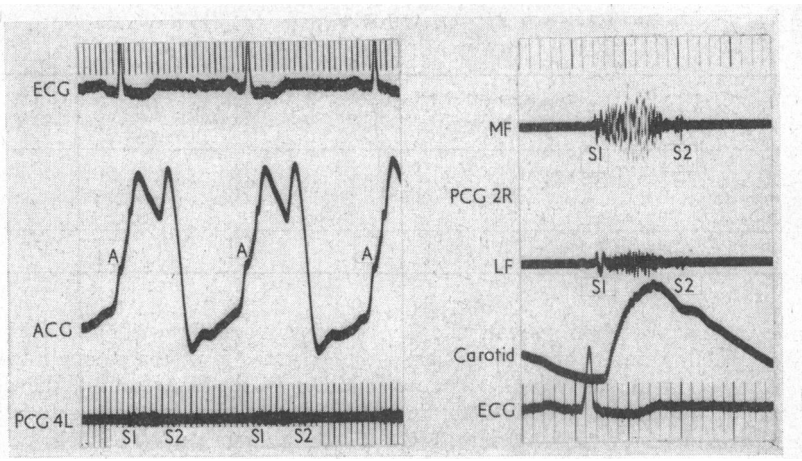

FIG. 11.—Patient No. 5. Aortic stenosis. Systolic gradient 75 mm. Hg. The apex cardiogram shows a large "A" wave and a double systolic impulse; a/H ratio 28 per cent. Two impulses were palpable at the apex. It was not possible to determine which two of the three systolic waves were palpable. The phonocardiogram shows a mid-systolic ejection murmur and an anacrotic pulse.

Rapid early filling may sometimes produce a palpable filling wave coincident with a third heart sound. This was largely confined in this series to patients with left ventricular failure.

Since palpation is a subjective technique we have attempted to minimize observer error by using the clinical findings of at least 2 observers. Allowing for possible errors, there was a good correlation between a tall "A" wave on the apex cardiogram and its palpability (Fig. 7). In all but 2 of our patients an a/H ratio of 14 per cent or more was associated with a palpable atrial impulse. One of these patients (No. 8) had a short P–R interval, with the "A" wave closely applied to the main systolic impulse (Fig. 10). The other patient (No. 62) had an abnormal thoracic wall with a virtually impalpable apex beat. Three patients had a palpable atrial impulse with a normal a/H ratio: 1 of them had a double systolic impulse and the first component may have been mistaken for a large "A" wave; we are unable to explain the normal a/H ratio in the other 2 patients.

Though large "A" waves can be detected by palpation, attempts at finer discrimination were unsuccessful and we could not reliably distinguish between moderate or marked increases in the size of the "A" wave.

Apex Cardiogram and Left Heart Pressures. There are few studies of left atrial pressure in normal subjects. Braunwald *et al.* (1961) studied 18 normal subjects and found an average left atrial peak "a" wave pressure of 10·4 mm. Hg (range: 4–16 mm. Hg) and an average "a" wave pulse pressure of 3·4 mm. Hg (range: 1–7 mm. Hg). Samet *et al.* (1965) found a left atrial peak "a" wave pressure in 32 normal subjects of 11 mm. Hg (range: 7–16 mm. Hg), using a lower zero manometer setting. In our experience it is unusual to find a normal left atrial peak "a" wave pressure exceeding 10 mm. Hg and an "a" wave pulse pressure of more than 3·5 mm. Hg. We therefore consider that a peak "a" wave pressure of 12 mm. Hg or more, and an "a" wave pulse pressure of more than 5 mm. Hg should both be regarded as abnormal.

Although left ventricular end-diastolic pressure and left atrial "a" wave pressure showed a tendency to rise as the a/H ratio increased, the correlation was poor (Fig. 2, 3, and 4). However, the apex cardiograms and clinical examination were performed with the patients resting quietly, whereas during catheter studies the patients were sedated and in a basal state. The different circulatory conditions in these two situations might explain an abnormal a/H ratio at the bedside and normal left heart pressures at catheterization. In fact 7 of 10 patients with a low resting peak left atrial "a" wave pressure and an increased a/H ratio showed a rise in left atrial pressure with the increase in cardiac output following angiography.

Simultaneous records of left heart pressures with the apex cardiogram show a positive correlation between the a/H ratio and the peak left atrial "a" wave pressure (Fig. 5). An a/H ratio of 14 per cent or more was always associated with a peak "a" wave pressure of at least 12 mm. Hg. When the a/H ratio was 20 per cent or more the peak "a" wave pressure exceeded 20 mm. Hg.

A group of 15 patients with severe left ventricular disease has been studied, consisting of 8

TABLE V
PALPABLE "A" WAVE AND FOURTH HEART SOUND

	Palpable "A" and fourth sound on auscultation	Palpable "A", no fourth sound	No "A" palpable, fourth sound on auscultation	No "A" palpable, no fourth sound
Aortic stenosis	10	5	1	13
Aortic stenosis and regurgitation	8	2	—	—
Aortic regurgitation	11	1	—	—
Cardiomyopathy	10	2	2	1
Total	39	10	3	14

patients from the main series of 66 patients and 7 additional patients. All were in obvious clinical left ventricular failure and had radiological evidence of pulmonary venous hypertension and therefore a mean left atrial pressure of at least 20 mm. Hg (Short, 1956; Selzer and McCaughey, 1960). Each patient had a palpable atrial impulse. The a/H ratio was 18 per cent or more in all but one patient and more than 20 per cent in 12 of the 15 patients. The findings in these 15 patients are similar to those shown by simultaneous records of left atrial pressure and the apex cardiogram, in that an a/H ratio of 20 per cent or more was associated with a peak left atrial "a" wave pressure of more than 20 mm. Hg (Fig. 5).

Other workers who have recorded the apex cardiogram and left heart pressures simultaneously have also found that an a/H ratio of 20 per cent or more was invariably associated with a left ventricular end-diastolic pressure of more than 20 mm. Hg (Dimond and Benchimol, 1963; Tavel *et al.*, 1965; Rios and Massumi, 1965). However, the converse did not apply and some patients had an a/H ratio of less than 20 per cent even though left ventricular end-diastolic pressure was greater than 20 mm. Hg.

These discrepancies between left atrial pressure and the a/H ratio remain unexplained. Some patients appear to have a large a/H ratio due to an increased left atrial stroke volume, with only a slight rise in left atrial pressure. Other patients have a high left atrial pressure without much increase in the a/H ratio or left atrial stroke volume. This does not invalidate the significance of a very large "A" wave on the apex cardiogram, and an a/H ratio of 20 per cent or more is always associated with a high left atrial pressure.

Relation of Atrial Impulse to Fourth Heart Sound. Table V compares the presence or absence of a palpable atrial impulse with the fourth heart sound on auscultation. Phonocardiographic studies showed occasional differences from the auscultatory findings. There were 49 patients with a palpable "A" wave, and 39 of these had an audible fourth heart sound. Only 3 patients had an audible fourth heart sound without a palpable atrial impulse. In 6 of the 10 patients with a palpable "A" wave and no audible fourth heart sound, the phonocardiogram did in fact demonstrate a fourth heart sound. These findings show the close relation that exists between the apical atrial impulse and the fourth heart sound. Benchimol and Dimond (1962) found a similar association in ischaemic heart disease. The same haemodynamic disturbance probably underlies both phenomena. The palpable "A" wave is direct evidence of a disturbed pressure-volume relation in the left ventricle. The fourth heart sound, on the other hand, bears a secondary relation to the haemodynamic disturbance, since there are many other factors that determine whether an audible sound develops.

It is of interest that Potain (1875), who described the presystolic (fourth) heart sound in hypertension, also noticed the accompanying presystolic atrial thrust by direct auscultation.

The atrial impulse is an easier physical sign to recognize than a fourth heart sound because of the low frequency of the sound. This especially applies in aortic stenosis when it may be difficult to distinguish between a fourth heart sound, a first heart sound, and a systolic click when all 3 are present (Fig. 12). A loud systolic murmur adds to this difficulty, since it impairs the ability of the ear to detect low-pitched sounds.

In aortic stenosis a large atrial impulse was of great value in assessing the severity of the obstruction, and our findings are similar to those of Tavel *et al.* (1965). If the a/H ratio was above 15 per cent or the atrial impulse was palpable, the systolic gradient was at least 75 mm. Hg, with only one exception (Fig. 13). These findings also agree closely with those of Goldblatt *et al.* (1962) and Caulfield, Perloff, and deLeon (1967), who attached the same haemodynamic significance to the presence of a fourth heart sound in aortic stenosis. The converse does not apply, since 8 patients had a peak systolic gradient of 75 mm. Hg or more, with a normal a/H ratio and without a palpable atrial impulse. Thus the absence of a large "A" wave on the apex cardiogram does not exclude severe aortic

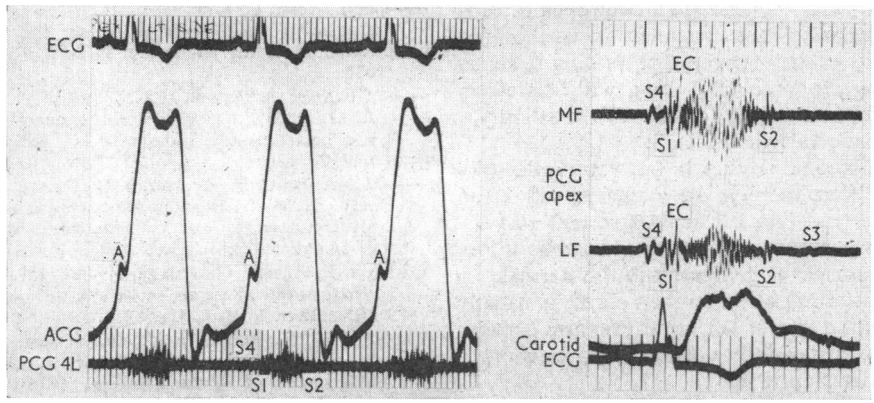

FIG. 12.—Patient No. 38. Aortic stenosis and regurgitation. Systolic gradient 110 mm. Hg. Palpable atrial impulse. a/H ratio 25 per cent. The phonocardiogram shows a fourth heart sound (S4) and a systolic click (EC). The carotid pulse is bisferiens in character. It was not possible to distinguish between S4, S1, and EC on auscultation, though two audible sounds preceded the systolic murmur.

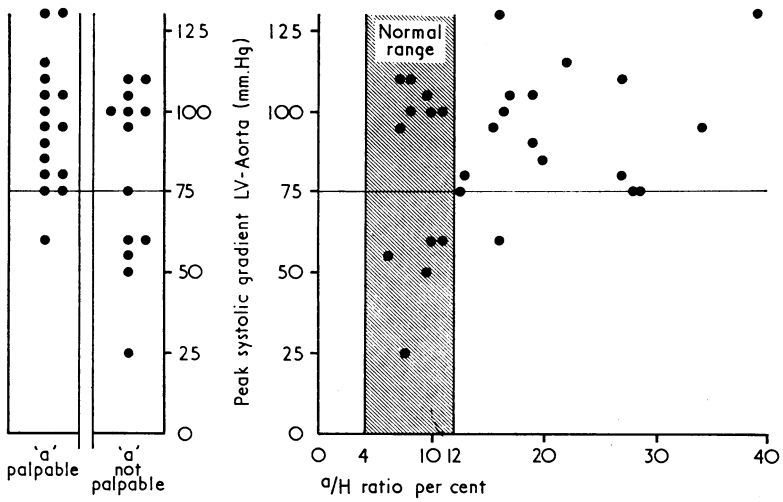

FIG. 13.—Twenty-nine patients with aortic stenosis. On the right the a/H ratio is plotted against the peak systolic gradient between left ventricle and aorta. An a/H ratio above normal was associated with a gradient of 75 mm. Hg or more with only one exception (Patient No. 26). On the left the gradient is compared with the presence of a palpable atrial impulse. A palpable atrial impulse was associated with a gradient of at least 75 mm. Hg with only one exception (Patient No. 26).

stenosis. This relation between the severity of the aortic stenosis and a large atrial impulse is indirect, since it reflects the greater disturbance of left ventricular function in more advanced disease.

A large atrial impulse was of great value in excluding a significant mitral valve lesion in patients with aortic valve disease. It helped in differentiating the Austin Flint apical diastolic rumble of aortic regurgitation from that of mitral stenosis. In our experience a palpable atrial beat excluded mitral stenosis of more than trivial degree.

SUMMARY

In left ventricular disorders tall "A" waves are frequently recorded on the apex cardiogram. They are due to displacement of the wall of the left ventricle in late diastole by left atrial systole.

If the apex cardiogram "A" wave is large, it can

usually be palpated. Although a large apex cardiogram "A" wave is most frequent in cardiomyopathy it is also a relatively common finding in aortic valve disease. A large "A" wave will, therefore, not differentiate between obstructive cardiomyopathy and aortic valve stenosis.

Cine-angiographic studies in left ventricular disorders show that there is an increased left atrial stroke volume and that left atrial systole is responsible for a larger proportion of the diastolic filling of the left ventricle as compared to the normal.

The increased left atrial stroke volume is usually associated with a rise of left atrial pressure because of the altered distensibility characteristics (compliance) of the left ventricle.

A positive relation exists between the size of the "A" wave on the apex cardiogram (a/H ratio) and the left atrial pressure. A large a/H ratio or a palpable atrial impulse implies an increase in the left atrial pressure above normal. An a/H ratio of 20 per cent was always associated with a peak left atrial "a" wave pressure of 20 mm. Hg or more.

A palpable atrial impulse is usually associated with a fourth (atrial) heart sound and both phenomena have similar haemodynamic significance.

In aortic stenosis a large a/H ratio or a palpable atrial impulse suggests that the stenosis is severe and that the systolic gradient is 75 mm. Hg or more. The converse does not apply.

A palpable atrial impulse in a patient with left ventricular disease excludes significant associated mitral stenosis.

The authors wish to thank Dr. E. Walker for providing radiological assistance. We are also grateful to Mrs. B. Lea, Senior Cardiographic Technician, and to Sister J. Davies, in charge of the Catheter Laboratory, and their respective staffs for invaluable technical help.

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